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Personal Exposure to Black Carbon at School and Levels of Fractional Exhaled Nitric Oxide in New York City

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Background: Schools are often located near traffic sources, leading to high levels of exposure to traffic-related air pollutants, including black carbon (BC). Thus, the school environment could play in a significant role in the adverse respiratory health of children.

Objectives: Our objective was to determine associations between personal BC levels at school and airway inflammation [i.e., fractional exhaled nitric oxide (FeNO)] in school-age children. We hypothesized that higher school BC (SBC) would be associated with higher FeNO.

Methods: Children 9–14 years of age in New York City (NYC) ($n = 114$) wore BC monitors for two 24-h periods over a 6-d sampling period, repeated 6 months later. SBC was defined as the average personal BC concentrations measured during NYC school hours (i.e., 0830–1430 hours). FeNO was measured following each 24-h BC monitoring period. Multivariable linear regression in generalized estimating equation models were used to examine associations between SBC and FeNO. Results are presented as percentage difference (PD) in FeNO.

Results: Personal BC at school was associated with higher FeNO (PD = 7.47% higher FeNO per 1-$\mu g/m^3$ BC (95% CI: 1.31, 13.9), $p = 0.02$]. Compared with BC exposure during school, a smaller PD in FeNO was observed in association with BC exposure while commuting to and from school [PD = 6.82% (95% CI: 0.70, 13.3), $p = 0.03$]. Personal BC in non-school environments and residential BC were not associated with FeNO ($p > 0.05$). A significant association between personal BC at school and FeNO was observed among children with seroatopy who did not have asthma [PD = 21.5% (95% CI: 4.81, 40.9), $p = 0.01$].

Discussion: Schools may be important sources of BC exposure that contribute to airway inflammation in school-age children. Our results provide rationale for interventions that target improved air quality in urban schools and classrooms. https://doi.org/10.1289/EHP8985

Introduction

Schools represent key environments where children spend a significant portion of their day. Thus, varying levels of air pollution exposures in the school environment could play a significant role in the respiratory health of children. For example, children who attend schools in close proximity to traffic sources are exposed to higher levels of traffic-related air pollution (TRAP), measured by either fixed school site monitors or personal monitors, compared with children whose schools are farther from major thruways (Boniardi et al. 2019; Spira-Cohen et al. 2010). Most studies that focus on children's exposure to air pollutants, particularly in school environments, have largely relied on exposure assessment using fixed monitoring sites (e.g., classroom or outside of school) (Flamant-Hulin et al. 2010; Jhun et al. 2017; Zora et al. 2013). Few studies have characterized school-age children's exposure to air pollutants via personal monitoring devices, which provide greater accuracy in defining variability as students travel to different classrooms, the cafeteria, gym period, and so on, and the ability to compare exposure levels in different environments such as home vs. school (Pañella et al. 2017; Paunescu et al. 2019; Spira-Cohen et al. 2010). Investigation of exposure to air pollution in school vs. non-school environments would help us to better understand the impact of school environments on children's health.

Exposure to air pollution, including black carbon (BC) or soot, a major component of fine particulate matter [PM $\leq$2.5 μm in aerodynamic diameter ($PM_{2.5}$)], has been associated with airway inflammation and increased asthma symptoms among school-age children (De Prins et al. 2014; Flamant-Hulin et al. 2010; Khreis et al. 2017; Paunescu et al. 2019). BC can be produced by any incomplete combustion source. Since the phase-out of residual fuel in New York City (NYC) in 2011, traffic is now a major source of BC (Jung et al. 2014); therefore, BC is commonly used as a surrogate for exposure to TRAP (HEI 2010). Epidemiological studies using BC as an indicator of TRAP reported that exposure to BC has been linked to reduced pulmonary function (Adam et al. 2015; Bowatte et al. 2017; Rice et al. 2015; Schultz et al. 2016) and increased airway inflammation (Berhane et al. 2014; Eckel et al. 2016; Wu et al. 2016). Furthermore, studies have suggested that BC is associated with worse health outcomes than $PM_{2.5}$ is when analyzed in two-pollutant models (Janssen et al. 2011; Lin et al. 2011).

Associations between BC exposure and elevated fractional exhaled nitric oxide (FeNO), a key noninvasive measure of allergic airway inflammation (Pijnenburg and De Jongste 2008; van der Valk et al. 2012), have been reported among school-age children (Cornell et al. 2012; De Prins et al. 2014; Lovinsky-Desir et al. 2016). Our group at the Columbia Center for Children's Environmental Health (CCCEH) showed that children who were exposed to higher levels of BC, assessed by 7-d integrated residential indoor home measurement, had increased levels of FeNO (Cornell et al. 2012). Another study, of a European birth cohort, found a significant association between BC levels, assessed with 24-h averaged personal measurement, and increases in FeNO among children with active respiratory symptoms (Paunescu et al. 2019). Yet these few studies have not addressed the school-specific exposures of BC and respiratory health outcomes.

School-age children living in urban communities like NYC may be at greater risk for elevated airway inflammation owing to

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their close proximity to roadways/highways (Patel et al. 2009). Our objective was to determine associations between personal BC levels at school, measured by personal monitoring devices, and airway inflammation (i.e., FeNO) in a nested cohort of school-age children with and without asthma living in NYC. Children with asthma or who are sensitized to cockroach allergen may be more susceptible to air pollution (Jung et al. 2017a; Lewis et al. 2005; Li et al. 2012). Therefore, we further examined whether the association between school BC (SBC) exposure and FeNO would differ by asthma phenotype (i.e., allergic asthma defined as sensitization to selected indoor and outdoor allergens). We hypothesized that higher BC exposure levels during school hours would be associated with increased FeNO and that the association between SBC and FeNO may vary by asthma/seroatopy.

Methods

Study Population and Personal Air Monitoring

Study participants ($n = 163$) were enrolled from the CCCEH longitudinal birth cohort (Jung et al. 2017a; Perera et al. 2002). For this nested observational study to evaluate environmental factors associated with respiratory outcomes (Jung et al. 2017b), children were recruited to meet criteria for age (9–14 y old) and current asthma status (target, 50% asthma, as diagnosed by a pulmonologist or allergist and a report of asthma symptoms or asthma medication use in the 1 y prior to enrollment) (Lovinsky-Desir et al. 2014). Maternal ethnicity and ethnicity of the child were determined by self-/parental-report. Children completed questionnaires at the end of each 24-h personal BC monitoring period to assess for the use of asthma controller medication [e.g., inhaled corticosteroids (ICS) and combined ICS-long acting beta agonists], time spent in each environment (e.g., school, home, others), cooking activities (yes/no), and current environmental tobacco smoke (ETS) exposure (i.e., the presence of smoker nearby) while wearing the personal monitor. Of 163 children, 145 had visits during times when they were in school. Complete data on SBC, FeNO, and serum immunoglobulin (Ig) E were available for $N = 114$ participants who were included in this analysis (Figure 1). Of the children with complete data, 98 had repeated measures 6 months later, as per the original study design. The study was approved by the Columbia University institutional review board (Human Subjects protocol AAAI0459), and written informed consents and assents were obtained.

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Personal Air Monitoring and Assessment of Air Pollution

Children wore a vest that contained a MicroAeth (Model AE51; Magee Scientific) personal BC monitor, which ran at 50 mL/min (Lovinsky-Desir et al. 2014). Personal BC levels were monitored every 5 min over two 24-h periods 5 d apart (BC_1, BC_2) and repeated 6 months later between March 2012 and August 2015 (Figure 2). All 5-min BC data were cleaned to remove false-positive and -negative data, as published previously (Cai et al. 2013, 2014), and averaged over each 24-h sampling period to obtain personal BC levels (Figure 2).

SBC was defined as the average personal BC concentrations (in micrograms per meter cubed) measured during NYC school hours (i.e., the 6 h between 0830 and 1430 hours) if children reported "Yes" to the question, "Did you go to school today?" (Figure 2). The average non-school BC (nonSBC) concentration was defined as

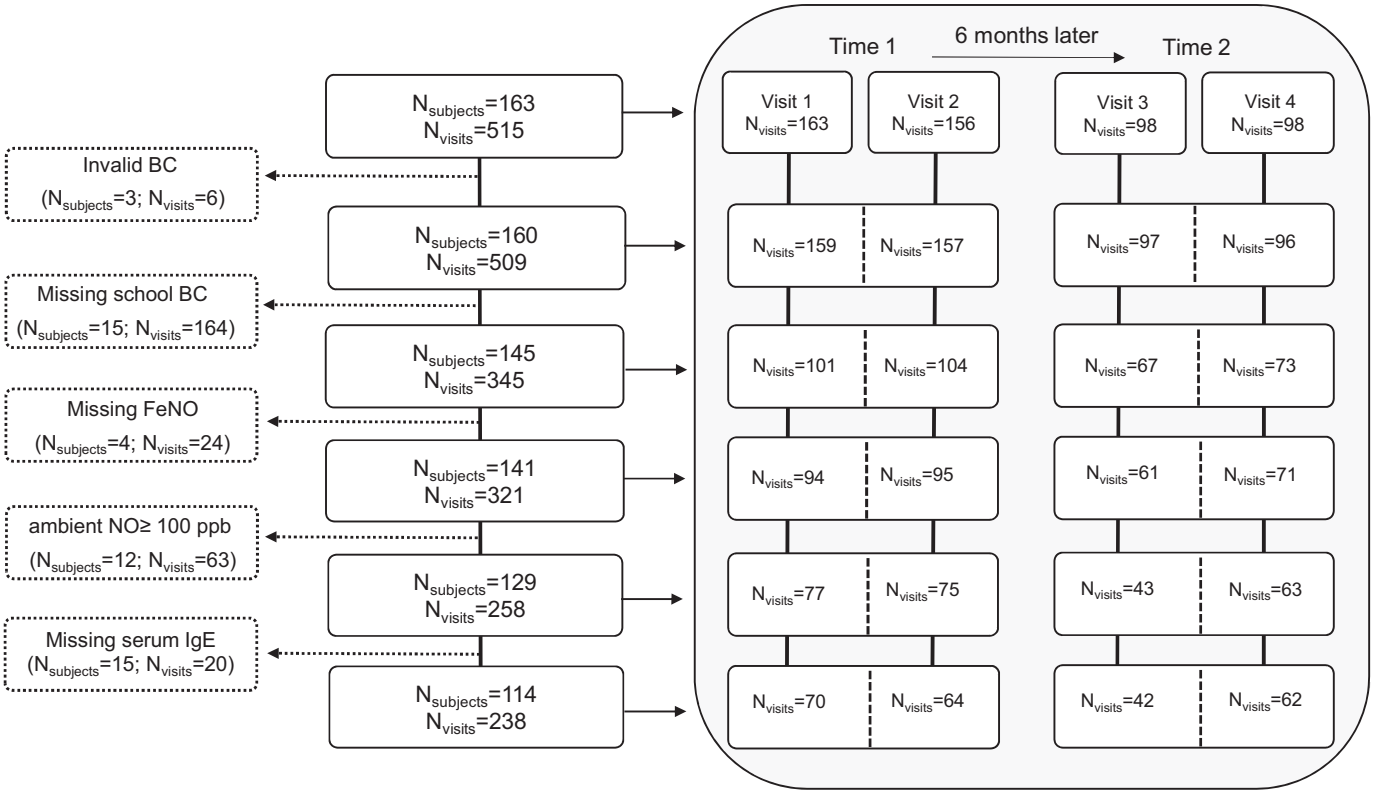


Figure 1. Schematic demonstration of study data structure. Complete data on SBC, FeNO, and serum IgE were available for $N = 114$ participants in NYC who were recruited between March 2012 and August 2015 and included in this analysis. Data collection was repeated 6 months later as Time 2. Note: BC, black carbon; FeNO, fractional exhaled nitric oxide; IgE, immunoglobulin E; NYC: New York City; SBC, school black carbon.

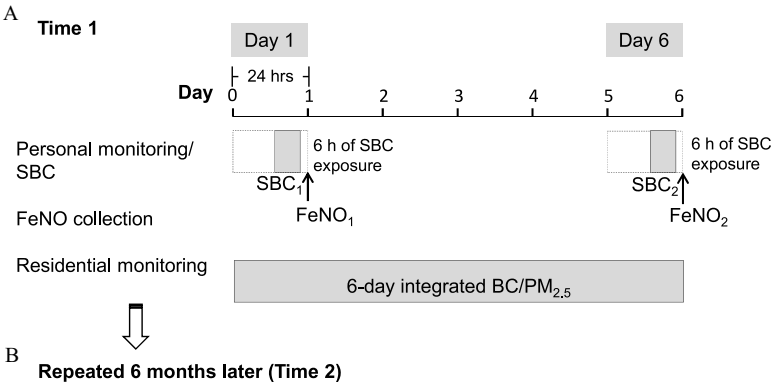


Figure 2. The study design for $N = 114$ schoolchildren in NYC recruited between March 2012 and August 2015. Personal BC, residential BC, and FeNO data collection at Time 1 and 2 are displayed. SBC was defined as the average personal BC levels measured between 0830 and 1430 hours (6 h; NYC public school hours); FeNO was collected at the end of each 24-h personal BC monitoring; residential $BC/PM_{2.5}$ levels were collected over 6 d, which overlapped with two 24-h personal BC monitoring periods. Data collection was repeated 6 months later, as Time 2. Note: BC, black carbon; FeNO, fractional exhaled nitric oxide; NYC: New York City; $PM_{2.5}$, fine particulate matter (PM $\leq$2.5 μm in aerodynamic diameter); SBC, school black carbon.

$$\text{NonSBC} = ([\text{24h-averaged personal BC concentration}, \mu g/m^3 \times 24\ h] - [\text{school BC concentration}, \mu g/m^3 \times 6\ h])/18\ h.$$

Using 5-min data, peaks of BC were defined if *a*) the PD (i.e., $[BC_i - BC_{i-1}]/BC_i \times 100$) between data point ($BC_i$) and the preceding data point (BC_{i-1}) was greater than 50%, and *b*) the PD between BC_i and the subsequent data point (BC_{i+1}), $[BC_{i+1} - BC_i]/BC_{i+1} \times 100$, was less than -50%.

Residential indoor BC and $PM_{2.5}$ were collected over 6 d that overlapped with the Day 1 and Day 6 personal sampling periods, as published previously (Jung et al. 2017b) (Figure 2). Indoor air monitors were placed in a room where the child spent most of his or her time (most often the room where the child slept).

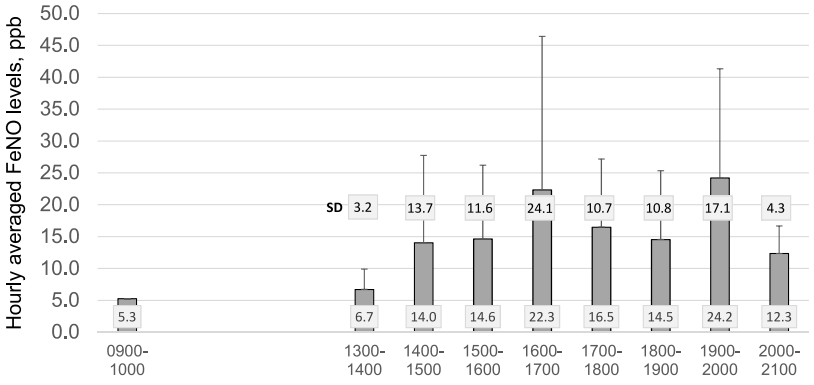
Allergic Sensitization

Total and specific IgE to indoor (German cockroach, mouse, cat, dog, and *Dermatophagoides farinae*) and outdoor allergens [common ragweed, mixed grass pollen (Gx2), and mixed tree pollen (Tx1)] were measured using Immunocap (Phadia) (Jung et al. 2015). Sera were collected at ages 7, 9, and 11 y as part of the birth cohort study, and analysis included data for the age closest to the child's current age. Common ragweed, mixed grass pollen, and mixed tree pollen data were not available for $n = 16$ children who were included in the study. Cat and dog allergens were not measured at 11 years of age. Children were classified as seroatopic if they had a specific IgE $\geq$0.35 IU/mL to any of the allergens tested.

Measurement of FeNO

FeNO was measured in the child's home on Day 1 ($FeNO_1$) and Day 6 ($FeNO_2$) following each 24-h personal BC collection, and the measurements were repeated 6 months later using the offline technique (GE Instruments) (Figure 2). The timing of FeNO measurement [mean $\pm$ standard deviation (SD)] was on average 2.4 ± 1.9 h after returning home from school (Figure 3). Three breath samples were collected in individual Mylar balloons at a flow rate of 83 mL/s (Rosa et al. 2014). Two ambient nitric oxide (NO) samples were simultaneously collected with FeNO using a NO analyzer (GE Instruments) to account for possible home environmental contamination of our sample. FeNO and ambient NO levels were respectively averaged across the two measures to obtain mean daily levels of FeNO and ambient NO.

A



B

Timing of FeNO measurement, hours	Frequency (%)
0900-1000	1 (0.4)
1300-1400	3 (1.3)
1400-1500	3 (1.3)
1500-1600	32 (13.4)
1600-1700	52 (21.8)
1700-1800	58 (24.4)
1800-1900	62 (26.1)
1900-2000	22 (9.2)
2000-2100	5 (2.1)
Total visits	238 (100)

Figure 3. (A) Averaged FeNO levels (ppb) measured at Time 1 Day 1 ($FeNO_1$) based on the time of day of measurement, and (B) the frequency of FeNO measurement within each time window, including repeat study visits. The bars represent the average values of FeNO levels measured within each time window and the lines represent standard deviations (SDs); numerical data for averages and SDs are presented in light gray boxes; 72.3% of the total FeNO measurements were taken between 1600 and 1900 hours. $N = 114$ participants in NYC who were recruited between March 2012 and August 2015. Note: FeNO, fractional exhaled nitric oxide; NYC: New York City.

Of $N_{visits} = 63$, 18% of FeNO samples were determined to be invalid owing to ambient NO >100 ppb, indicating potential home environmental contamination (Cornell et al. 2012) (Figure 1).

Data Analysis

Analyses were restricted to children who had complete data on SBC, FeNO, ambient NO, and seroatopy for at least one study visit, resulting in a final sample number of 114 (Figure 1). In comparison with 6-d integrated residential BC, the average of the first two measurements of SBC (SBC_1 and SBC_2) and FeNO ($FeNO_1$ and $FeNO_2$) at Time 1 were used for descriptive statistics (Figure 2). Descriptive statistics were analyzed using Mann-Whitney tests, Wilcoxon–rank sum tests, and Spearman correlations, as appropriate. Because of the nonnormal distribution of FeNO, log-transformed FeNO levels were used for subsequent analyses.

Associations between SBC levels and FeNO were analyzed using multivariable linear regression in generalized estimating equation models, with robust standard errors, controlling for covariates that are known to influence both BC and FeNO. Two types of models were applied: *a*) basic models with an adjustment for ambient NO only (the basic model), and *b*) fully adjusted models with adjustment for asthma diagnosis, age, current ETS exposure, three dummy variables for season (summer, fall and winter), ambient NO, and the hour of FeNO measurement (the fully adjusted model). A <10% change in the estimate was observed when we included seroatopy and biological sex in main models (Table S1), thus, to maintain the most parsimonious models, seroatopy and sex were excluded from the final models. In the fully adjusted model used to assess the relationship between nonSBC and FeNO (the nonSBC model), we further controlled for cooking activities at home because cooking emissions can be a major source of indoor BC (Jeong et al. 2019). In the fully adjusted model used to assess the relationship between residential BC (6-d integrated) and FeNO (the average between $FeNO_1$ and $FeNO_2$ (the residential BC model), we controlled for the average time spent at home, cooking activities, and the aforementioned covariates.

To explore the influence of transportation/commuting exposure on FeNO, we estimated the commute BC exposure levels based on the average personal BC exposure measured 1 h before (0730–0830 hours) and after school hours (1430–1530 hours) and then examined the association between commute BC exposure and FeNO in the fully adjusted model.

To further assess whether the association between SBC and FeNO varied by asthma/seroatopy, the fully adjusted model was stratified by asthma and seroatopy (i.e., four groups: no asthma/no seroatopy, no asthma/seroatopy, asthma/no seroatopy, and asthma/seroatopy). To test whether the association between SBC and FeNO varied by asthma phenotype, a three-way multiplicative interaction term (SBC × seroatopy × asthma diagnosis) was included in the fully adjusted model along with seroatopy and the two-way interaction terms (SBC × seroatopy, SBC × asthma diagnosis, and seroatopy × asthma diagnosis). The results are presented as percentage difference (PD) in FeNO, which was calculated as $100 \times [\exp(\beta_{adj}) - 1]$ where β_{adj} is a coefficient of BC in the log-transformed FeNO in adjusted models.

Sensitivity analyses were conducted in the fully adjusted model as follows: *a*) reanalysis including only children who had personal BC levels during school hours on both school days and non-school days ($N_{subjects} = 50$); *b*) reanalysis after controlling for residential $PM_{2.5}$ and nonSBC; *c*) reanalysis after excluding children who had extreme FeNO values, >90th percentile (38 ppb; $N_{subjects} = 8$, $n_{visits} = 24$); *d*) reanalysis after removing the upper and lower 5% of SBC measures; *e*) reanalysis after removing children whose FeNO was measured during school hours ($N_{subjects} = 4$, $n_{visits} = 4$); *f*) reanalysis after controlling for any report of asthma controller medication (yes/no); and *g*) reanalysis after redefining school hours as between 0900 and 1400 hours to remove potential misclassification of exposure that may have included transportation/commute exposure given the observed associations between BC exposure during commutes and increased FeNO in our previous study (Lovinsky-Desir et al. 2019). All analyses were performed using SPSS (version 25; IBM SPSS), where $p < 0.05$ was considered statistically significant.

Results

Cohort Characteristics

Characteristics of the 114 children are presented in Table 1. Based on recruitment strategy for the nested parent study, there was a large proportion of children with asthma (54%, $N = 62$) and seroatopy (62%, $N = 71$). The children attended 85 different schools in total, with $N = 68$ (61.2%) children who went to unique schools having no overlap with other children (Table S2).

Personal SBC Levels and Peak Exposures

At Time 1, the median [interquartile range (IQR)] of BC at school was 0.99 (1.01) $\mu g/m^3$ and SBC levels were not significantly different from nonSBC [Table S3A; median (IQR) = 1.16 (1.10) $\mu g/m^3$]. In contrast, children experienced more frequent peaks or variability in BC exposure per hour during school hours compared with non-school hours [Table S3B; median (IQR) = 0.33 (0.50) vs. 0.17 (0.22) during school and non-school hours, respectively, $p < 0.01$]. The estimated 2-h averaged commute BC levels [Table S3A; median (IQR) = 1.31 (1.09) $\mu g/m^3$] were significantly higher than SBC exposure levels ($p < 0.01$).

The levels of hourly personal BC during school hours ranged between 1.26 and 1.66 $\mu g/m^3$. There were higher mean personal BC concentrations before (0700–0800 hours) and after school hours (1500–1600 hours), whereas the highest concentrations were observed between 1900 and 2100 hours (Figure 4). At Time 1, averaged SBC levels across the two 24-h sampling periods that overlapped with residential monitoring were moderately correlated with nonSBC (Figure S1; $r = 0.61$, $p < 0.01$) but not with the 6-d integrated residential indoor BC from the same children (Figure S1; $r = 0.07$, $p = 0.48$). Commute BC levels were moderately correlated with SBC ($r = 0.68$, $p < 0.01$) but minimally correlated with residential indoor BC ($r = 0.19$, $p = 0.05$). In comparison, nonSBC was moderately correlated with residential indoor BC (Figure S1; $r = 0.47$, $p < 0.01$). On individual days of sampling, SBC was moderately correlated with nonSBC ($r = 0.51$–0.54, $p < 0.01$).

Associations between Personal SBC Exposure and FeNO Levels

At Time 1, the median level of FeNO was 13.0 ppb (IQR: 11.1). The majority of FeNO [$N_{visits} = 172/238$ (72%)] measurements were taken between 1600 and 1900 hours, whereas $N_{visits} = 4$ (1.7%; $N_{subjects} = 4$) were measured during school hours (Figure 3). The highest FeNO concentration was observed between 1900 and 2000 hours, followed by the period 1600–1700 hours (Figure 3). For $N_{visits} = 57$ (23.9%, $N_{subjects} = 32$), FeNO levels were greater than the 20-ppb "normal" threshold for children identified by the American Thoracic Society. Children with asthma exhibited significantly higher levels of FeNO compared with children with no asthma [median (IQR) = 14.5 (15.3) vs. 12.2 (9.6) ppb for asthma vs. no asthma, $p = 0.03$]. Similarly, children with seroatopy had higher levels of FeNO than children without seroatopy [median (IQR) = 14.4 (12.8) vs. 9.3 (7.8) ppb for seroatopy vs. no seroatopy, $p < 0.01$].

Table 1. Cohort characteristics of schoolchildren ($N = 114$) in NYC recruited between March 2012 and August 2015.

Characteristic	Participants included[a] ($N = 114$)
Maternal ethnicity [n (%)]	
Dominican	70 (61.4)
African American	44 (38.6)
Age {y [mean (min-max)]}	12.8 (10.4–14.3)
Sex [n (%)]	
Girls	63 (55.3)
Boys	51 (44.7)
Maternal education [n (%)]	
<High school degree	45 (41.3)
≥High school degree	64 (58.7)
Missing [n]	5
Maternal asthma [n (%)]	
Yes	31 (27.2)
No	83 (72.8)
Current ETS exposure [n (%)][b]	
Yes	42 (36.8)
No	72 (63.2)
On controller medication[c]	
Yes	5 (4.4)
No	109 (95.6)
NYC school type [n (%)]	
NYC public schools	104 (95.4)
Private/religious	5 (4.6)
Missing [n]	5
Daily time spent at home [h (mean ± SD)][d]	15.8 ± 3.07
Cooking activities [n (%)][e]	
Yes	91 (79.8)
No	23 (20.2)
Sampling season[f]	
Spring (March 20–June 19)	43 (37.7)
Summer (June 20–September 9/21)	28 (24.6)
Fall (September 22–December 20)	20 (17.5)
Winter (December 21–March 19)	23 (20.2)
Residential $PM_{2.5}$ [$\mu g/m^3$ (mean ± SD)][g]	14.2 ± 9.1
Missing [n]	5
BMI [z-score (mean ± SD)][h]	0.86 ± 1.11
Obesity {≥95th percentile [n (%)]}	
Yes	28 (24.6)
No	86 (75.4)
Asthma [n (%)][i]	
Yes	62 (54.4)
No	52 (45.6)
Seroatopy [n (%)][j]	
Yes	71 (62.3)
No	43 (37.7)
Total IgE [IU/mL (mean ± SD)]	290.5 ± 660.3
Ambient NO {ppb [median (IQR)]}[k]	19.2 (34.5)

Note: BMI, Body mass index; ETS, environmental tobacco smoke; FeNO, fractional exhaled nitric oxide; IgE, immunoglobulin E; IQR, interquartile range; max, maximum; min, minimum; NO, nitric oxide; NYC, New York City; $PM_{2.5}$], fine particulate matter (PM ≤2.5 μm in aerodynamic diameter); SD, standard deviation; SBC, school black carbon.
[a]Included only the children who had complete SBC, FeNO, and serum IgE data for the present analysis.
[b]Report of any smoker nearby during 24-h sampling period at either Day 1 and Day 6 in Time 1.
[c]Any controller medication reported in the past 24 h at either Day 1 and Day 6 in Time 1.
[d]Average time spent at home, determined by 24-h questionnaire administrated on both Day 1 and Day 6 in Time 1.
[e]Report of cooking activities during 24-h sampling period at either Day 1 or Day 6 in Time 1.
[f]Sampling season based on the first set-up date (Time 1).
[g]6-d integrated residential indoor $PM_{2.5}$ concentrations at Time 1.
[h]Weight (kg)/height $(m)^2$.
[i]Determined by a pulmonologist or allergist, to meet enrollment criteria for the nested study, children had to report having symptoms or using asthma medication in the 1 y prior to recruitment.
[j]Specific IgE ≥0.35 IU/mL to German cockroach, mouse, cat, dog, *Dermatophagoides farina*, common ragweed, mixed grass pollen (Gx2), or mixed tree pollen (Tx1).
[k]Average ambient NO levels measured at Time 1 (Day 1 and Day 6).

In models without covariate adjustment except ambient NO concentration (i.e. the basic model), personal BC levels at school were associated with higher FeNO [Table 2; PD = 8.33% higher in FeNO per 1-$\mu g/m^3$ BC (95% confidence interval (CI): 1.82, 15.4), $p = 0.01$]. After controlling for covariates (fully adjusted model), a significant association was observed between personal SBC and FeNO [Table 2; PD = 7.47% (95% CI: 1.31, 13.9), $p = 0.02$]. An association was observed between BC during commuting hours and FeNO [Table 2; PD = 6.82% (95% CI: 0.70, 13.3), $p = 0.03$], although this PD was smaller than observed between SBC and FeNO. In comparison, neither non-school personal BC nor 6-d integrated residential BC levels were associated with FeNO (Table 2).

In analyses stratified by asthma/seroatopy, a significant association between personal SBC level and FeNO was observed among seroatopic children who did not have asthma [Figure 5; Table S4; PD = 21.5% (95% CI: 4.81, 40.9), $p = 0.01$]. We did not observe significant associations between SBC and FeNO in the other subgroups (Figure 5; Table S4). The interaction between SBC and FeNO by asthma and seroatopy was not significant ($p_{interaction} = 0.45$).

Sensitivity Analyses

The results of the sensitivity analyses are shown in Table 3. First, in the restricted analysis among those who had personal BC levels during school hours on both school days and non-school days ($N_{subjects} = 50$), there was no association between personal BC levels (0830–1430 hours) and FeNO on non-school days [$N_{visits} = 72$, PD = −0.60% (95% CI: −9.15, 8.65), $p = 0.89$]. However, on school days, in this subgroup of 50 children, a positive but nonstatistically significant association between BC during school hours and FeNO was observed [$N_{visits} = 85$, PD = 9.64% (95% CI: −0.60, 20.9), $p = 0.07$]. Second, after adjustment for both residential $PM_{2.5}$ and nonSBC levels, significant associations between personal SBC and FeNO were replicated [PD = 7.47% (95% CI: 1.21, 14.1), $p = 0.02$]. Third, when children who had FeNO >38 ppb (90th percentile) were removed, a similar PD was noted [PD = 7.68% (95% CI: 2.53, 13.0); $p < 0.01$]. Fourth, after removing the top 5% of SBC measures, the association between SBC and FeNO remained significant [PD = 12.2% (95% CI: 2.63, 22.5); $p = 0.01$]. After removing the bottom 5% of SBC measures, the association between SBC and FeNO also remained significant [PD = 6.72% (95% CI: 0.20, 13.7); $p = 0.04$]. Fifth, when children whose FeNO measurements were taken during school hours were removed, a similar PD was noted [PD = 7.04% (95% CI: 0.90, 13.5); $p = 0.03$]. Sixth, after adjusting for asthma controller medication, significant associations between SBC and FeNO were replicated [PD = 7.25% (95% CI: 1.11, 13.8); $p = 0.02$]. Last, after redefining SBC exposure as 0900–1400 hours, a similar association was obtained between SBC and FeNO [PD = 7.25% (95% CI: 0.80, 14.0); $p = 0.03$].

Discussion

Our objective was to assess associations between personal BC exposure measured in the school environment and airway inflammation. In our sample of 114 children living in NYC, we observed a positive association between personal exposure to BC at school and FeNO, but no association between personal non-school or residential BC and FeNO, except during commuting to and from school. Our results highlight that air pollution exposure in school environments may be an important contributor to airway inflammation in schoolchildren. Furthermore, we observed that the association between SBC exposure and FeNO was significant only among seroatopic children without asthma. Our findings offer rationale for targeted interventions to improve air quality, particularly in schools, where children spend at least 25%

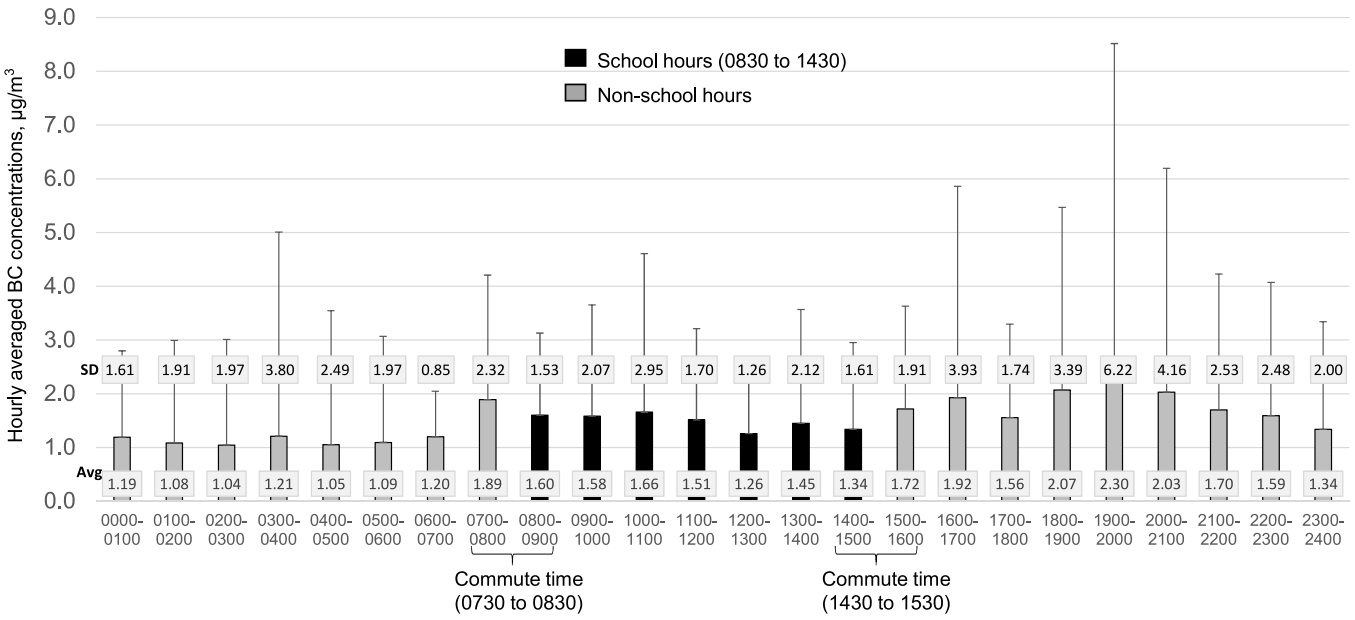


Figure 4. Averaged personal BC concentrations ($\mu g/m^3$) at Time 1 Day 1 based on the time of day of the measurement. All 5-min BC data were averaged hourly. The bars represent the averages and the lines represent standard deviations (SDs); numerical data for averages and SDs are presented in light gray boxes. NYC public school hours: 0830–1430 hours (6 h); non-school hours: 1430–0830 hours (18 h); estimated commute time: 0730–0830 hours and 1430–1530 hours (2 h). $N = 114$ participants in NYC who were recruited between March 2012 and August 2015. Note: BC, black carbon; NYC, New York City.

of their daily hours (e.g., 6 h of NYC public school hours) during the school year.

In the present study, we focused on BC exposures measured during NYC public school hours rather than 24-h averaged BC, using a real-time personal BC monitor. Most studies that focused on school environments have used measurements from school-based fixed monitoring sites (e.g., classroom, outside of school) (Flamant-Hulin et al. 2010; Jhun et al. 2017; Patel et al. 2013; Zora et al. 2013). More recently, studies using similar real-time personal monitoring devices with schoolchildren have characterized BC exposure levels in different environments (e.g., home, school, transportation) (Jeong and Park 2017, 2018; Pañella et al. 2017; Rivas et al. 2016). Although those studies highlighted each environment's contribution to overall daily BC exposure, they did not further link BC exposure in each environment to respiratory health outcomes. In our previous adult study that focused on transportation/commuting environments as a major contributor of BC exposure, we demonstrated that exposure to BC during a long commute was significantly higher compared with during noncommute times (Lovinsky-Desir et al. 2019). In addition, higher evening commute BC exposures were associated with higher FeNO the following morning (Lovinsky-Desir et al. 2019). Our application of real-time personal monitors combined with respiratory outcomes enabled us to discern SBC vs. nonSBC exposure levels at an individual level and to assess the specific role of SBC exposure on airway inflammation. Schoolchildren could receive the most intense exposure to BC during transportation (Jeong and Park 2017). Although the hourly averaged personal BC exposure was most elevated in the evenings, possibly attributed to home cooking activities, there was also a pattern of elevation in BC before and after school hours, possibly owing to commuting to school. Although we were not able to capture the exact periods of commute time, the additional analysis on the estimated commute BC exposure demonstrated that the levels of commute BC exposure were significantly higher than those during SBC. In addition, BC exposure during commuting hours was associated with FeNO, although perhaps not as strongly as the observed association between SBC and FeNO. To further assess

Table 2. Main effect associations [($N_{subjects}$:n_{visits} = 114:238)] between personal BC and FeNO levels in NYC schoolchildren ($N = 114$) recruited between March 2012 and August 2015.

	Basic model[a]			Fully adjusted model[b]		
BC exposure	Percentage difference (%)	95% CI	*p*-value	Percentage difference (%)	95% CI	*p*-value
SBC[c]	8.33	1.82, 15.4	0.01	7.47	1.31, 13.9	0.02
Commute BC[d]	6.18	0.60, 12.1	0.03	6.82	0.70, 13.3	0.03
NonSBC[e]	2.22	−0.50, 5.13	0.12	1.92	−1.09, 5.13	0.22
Residential BC[f]	−1.98	−12.2, 9.42	0.72	−0.50	−9.88, 9.75	0.92

Note: Percentage difference in FeNO are presented per 1-$\mu g/m^3$ increase in BC. Multivariable linear regression in generalized estimating equation models with robust standard errors were used to determine *p*-values to compare percentage difference in FeNO per 1-$\mu g/m^3$ increase in BC concentration. BC, black carbon; CI, confidence interval; ETS, environmental tobacco smoke; FeNO, fractional exhaled nitric oxide; NO, nitric oxide; nonSBC, non-school black carbon; $N_{subjects}$, number of subjects included for the analysis; n_{visits}, total number of visits; NYC, New York City; SBC, school black carbon.

[a]Basic models adjusted for ambient NO only.

[b]Models adjusted for asthma, age, current ETS exposure, three season dummy variables (summer, fall, and winter), ambient NO, the hour of each FeNO measurement, cooking activities (nonSBC and residential BC models only), and daily time spent at home (residential BC model only).

[c]Average personal BC exposure levels measured during NYC public school hours (0830–1430 hours (6 h)].

[d]Estimated average personal BC exposure levels measured from 0730 to 0830 hours and from 1430 to 1530 hours (2 h).

[e]Average personal BC exposure levels measured during non-school hours [1430–0830 hours (18 h)].

[f]6-d integrated residential BC exposure; $N_{subjects}$:n_{visits} = 111:176; three participants had missing residential BC data.

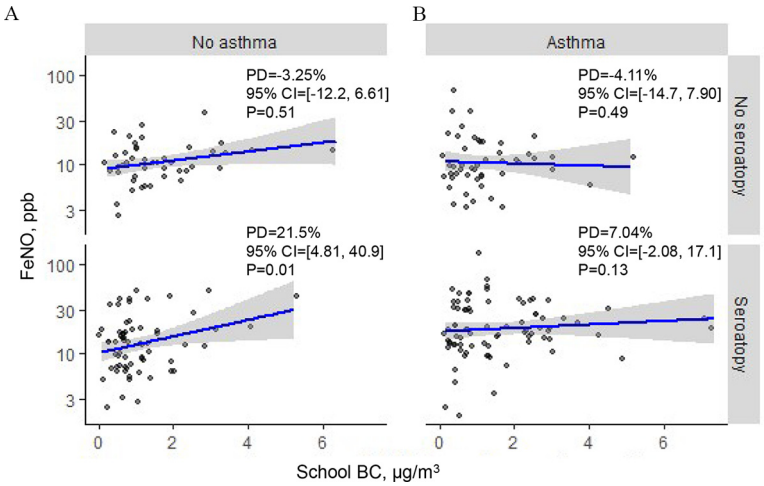


Figure 5. Associations between SBC and FeNO by asthma phenotype: (A) no asthma, (B) asthma. The regression lines with 95% CIs represent a univariate analysis. Presented values of percentage difference (PD), 95% CIs, and *p*-values were obtained from fully adjusted models (controlling for age, current ETS exposure, season dummy variables, ambient NO, and the hour of FeNO measurement). The interaction between SBC and FeNO by asthma and seroatopy was not significant ($p_{interaction} = 0.45$). $N = 114$ participants in NYC who were recruited between March 2012 and August 2015. Note: BC, black carbon; CI, confidence interval; ETS, environmental tobacco smoke; FeNO, fractional exhaled nitric oxide; NO, nitric oxide; NYC, New York City; SBC, school black carbon.

whether SBC–FeNO associations were attributed to exposure from transportation/commuting, we repeated the main analysis after removing 60 min of BC exposure from school hours (0900–1400 hours) in order to minimize potential contamination by transportation/commute-related BC exposure. The results remained significant, suggesting that personal BC exposure at school contributes to the higher FeNO levels in children. Nevertheless, the moderate correlation between SBC and commute BC exposure, as well as a similar association with FeNO, suggests that BC exposure while commuting to and from school could also contribute to the observed associations between SBC and FeNO.

Interestingly, although BC exposure at school was more strongly associated with FeNO, averaged BC exposure levels at school were not significantly different from those during non-school hours. There are several plausible explanations for these differences. For one, we observed that children were exposed to a greater number of peaks per hour during school hours than during non-school hours. Recent studies have focused on the characteristics of peaks, such as the number of peaks per hour, duration of peaks, or average concentration within a peak instead of the averaged air pollution exposure over the entire sampling period (Dons et al. 2019; Jeong and Park 2018). Observations reveal that the occurrence of peaks change over time, based on daily activities and environments. Schools in urban communities, such as NYC, are often located in close proximity to roadways/highways (Kingsley et al. 2014). Indeed, a study of TRAP levels in NYC found that a greater number of trucks/buses per hour was associated with an increase in BC, suggesting that diesel traffic emissions may be important sources of BC in NYC schools (Patel et al. 2009). Further studies to link the characteristics of peaks and health outcomes are needed to gain a better understanding of health relevance of peaks in pollution exposures. Second, physical activity/exercise at school may increase minute ventilation and could result in increased lung deposition of pollutant gases and particles (Löndahl et al. 2007). This was corroborated by our previous findings that increased outdoor activity in an urban environment was associated with lower lung function during warmer months (Lovinsky-Desir et al. 2021) and our unpublished observations that children are more active on school days than non-school days. Third, poor ventilation systems in older school buildings may place children at greater risk owing to

Table 3. Sensitivity analyses of associations between personal SBC exposure and FeNO levels in NYC schoolchildren ($N = 114$) recruited between March 2012 and August 2015.

		Fully adjusted[a]		
Sensitivity analyses	$N_{subjects}$:n_{visits}	Percentage difference (%)	95% CI	*p*-Value
1a. With children who had personal BC levels during school hours on both school days and non-school days: school hours on school days	50:85	9.64	−0.61, 20.9	0.07
1b. School hours on non-school days	50:72	−0.60	−9.15, 8.65	0.89
2. With an adjustment for both residential $PM_{2.5}$ and nonSBC levels	108:224	7.47	1.21, 14.1	0.02
3. After removing children who had FeNO >38 ppb (90th percentile)	106:214	7.68	2.53, 13.0	<0.01
4a. After removing the top 5% of SBC measures	111:227	12.2	2.63, 22.5	0.01
4b. After removing the bottom 5% of SBC measures	113:227	6.72	0.20, 13.7	0.04
5. After removing children whose FeNO measurements were taken during school hours	113:234	7.04	0.90, 13.5	0.03
6. With an adjustment for asthma controller medication	114:238	7.25	1.11, 13.8	0.02
7. After redefining SBC exposure (0900–1400 hours)	114:238	7.25	0.80, 14.0	0.03

Note: Percentage differences in FeNO are presented per 1-$\mu g/m^3$ increase in BC. Multivariable linear regression in generalized estimating equation models with robust standard errors were used to determine *p*-values to compare percentage difference in FeNO for 1-$\mu g/m^3$ increase in BC concentration. BC, black carbon; CI, confidence interval; ETS, environmental tobacco smoke; FeNO, fractional exhaled nitric oxide; NO, nitric oxide; nonSBC, non-school black carbon; $N_{subjects}$, number of subjects included for the analysis; n_{visits}, total number of visits; NYC, New York City; $PM_{2.5}$], fine particulate matter (PM ≤2.5 µm in aerodynamic diameter); SBC, school black carbon.
[a]Models were fully adjusted for age, asthma, current ETS exposure, three season dummy variables (summer, fall, and winter), ambient NO, and the hour of FeNO measurement..

the building up of air pollutants from indoor sources, such as heating (Fisk 2017). Ventilation rates in classrooms often did not meet the minimum standards set by the American Society of Heating, Refrigerating, and Air Conditioning Engineers and the European Committee for Standardization, and increased school ventilation rates are associated with reduced respiratory health (Fisk 2017).

Our results are consistent with those of other studies that found associations of BC with FeNO in general populations of schoolchildren (Cornell et al. 2012; De Prins et al. 2014). For example, in a study of 7- to 8-y-old children in NYC, Cornell et al.(2012) found a positive association between FeNO and BC when BC exposure was measured via 7 d of fixed monitoring within participants' homes. Similarly, in a study of 6- to 12-y-old children living in Belgium (De Prins et al. 2014), IQR increase in BC, either 24-h averaged BC at a central monitoring site or weekly BC concentration estimated via land-use regression modeling, was positively associated with FeNO. To our knowledge, our findings provide the first evidence that personal BC exposure measured specifically at school, as opposed to non-school environments, is associated with increased FeNO in schoolchildren living in an urban community. This result suggests that schools are an important environment when assessing exposure to BC and its association with airway inflammation.

Recent studies that investigated differing lag times have consistently shown the associations between the relatively short lag of BC/diesel exhaust and FeNO (Barath et al. 2013; Ji et al. 2021). For example, a study of children with asthma reported a significant association between BC and FeNO at lag periods of <12 h but not at longer lag times, including 12–24 h (Ji et al. 2021). Similarly, in a randomized controlled experimental exposure study of healthy human adult participants, exposure to diesel exhaust increased FeNO at 6 h, with diminished effect at 24 h (Barath et al. 2013). In the present study, because 72.3% of FeNO measurements were collected between 1600 and 1900 hours, it is possible that the observed association between SBC and FeNO could be attributed to our study design that assessed FeNO shortly after children returned from school. Thus, one explanation for the observed association between FeNO and SBC, but not with nonSBC, could be due to the timing of FeNO measurement rather than its causal importance. Although adjustment for the hour of FeNO measurement did not impact the observed association between BC and FeNO, it remains possible that the different lags between BC exposure and FeNO measurements may still explain some of the observed differences. In addition, when we repeated the main analysis with $N = 50$ children who had personal BC exposure levels during school hours on both school days and non-school days, a similar but nonsignificant association was observed between SBC and FeNO. However, during the same period of time on non-school days, there was no association between personal BC exposure during typical school hours and FeNO. This sensitivity analysis supports our hypothesis of a unique association between BC exposures experienced during the school day and FeNO.

Although children with asthma are often described as having greater airway inflammation, several studies have demonstrated that even children without asthma are at increased risk for airway inflammation in response to environmental pollution exposure (Flamant-Hulin et al. 2010; Patel et al. 2013). For example, in a panel study of adolescents both with and without asthma in NYC, increases in airway inflammation, measured by exhaled breath condensate pH, was associated with traffic-related air pollutants regardless of asthma diagnosis (Patel et al. 2013). In addition, in a controlled chamber exposure study, healthy adults exhibited increased airway inflammation following 2-h diesel exhaust particle exposure [100 $\mu g/m^3$ PM ≤ 10 μm in aerodynamic diameter (PM_{10})], whereas participants with asthma did not show any changes, suggesting that increased sensitivity in adults with asthma to TRAP is not necessarily associated with allergic asthmatic inflammation (Behndig et al. 2011). In the present study, the personal SBC and FeNO relationship was significant only among seroatopic children who did not have asthma. In fact, among children with atopy who did not have asthma, the 21.5% difference in FeNO that we observed with a 1-$\mu g/m^3$ increase in BC exposure reaches the threshold of clinically significant change in FeNO as indicated by the American Thoracic Society guidelines (Dweik et al. 2011). Our findings are comparable with a study of French children that demonstrated that children without asthma had a greater increase in FeNO in association with high $PM_{2.5}$ levels in classrooms when compared with children with asthma (Flamant-Hulin et al. 2010). Further, in their subgroup analysis of children without asthma, children with seroatopy had a stronger association between $PM_{2.5}$ and higher levels of FeNO compared with children without seroatopy (Flamant-Hulin et al. 2010). Taken together, our findings suggest that even children who do not have clinically diagnosed asthma are at risk for respiratory complications from environmental air pollution exposure.

The mechanisms underlying the association between BC and airway inflammation may involve environmental epigenetic regulations, such as DNA methylation. For example, exposure to BC has been associated with altered asthma gene DNA methylation in mouse experiments (Niedzwiecki et al. 2012), as well as in elderly adults (Sofer et al. 2013) and in children (Brunst et al. 2013). In addition, our previous study demonstrated that 24-h averaged BC was associated with the demethylation of one asthma gene, interleukin 4 (*IL4*), and the associations of BC with lowering *IL4* and induced nitric oxide synthase (encoded by *NOS2A*) methylation appeared to be stronger among the seroatopic children (Jung et al. 2017a). Further, the same study found that demethylation of *IL4* and *NOS2A* was associated with elevated FeNO, suggesting that BC may induce allergic asthma pro-inflammatory gene demethylation that in turn may link to airway inflammation (Jung et al. 2017a). In addition, diesel exhaust particle (DEP) exposure produces reactive oxygen species in the lungs and the oxidative stress induced by DEP exposure can lead to a cascade of reactions, including cell signaling by kinases, transcription factor activation, and inflammatory mediator release (Ghio et al. 2012).

We acknowledge several limitations. First, personal SBC levels were estimated based on the NYC public school hours instead of actual time spent within school buildings, leading to potential measurement error in SBC concentrations. Second, the sample size was fairly small, especially for the analyses stratified by asthma phenotype. Third, within the study design, we were not able to distinguish the sources of SBC and nonSBC exposure. However, given the moderate correlation between nonSBC and residential BC, the considerable time spent at home during the sampling period (68%), and the highest hourly mean personal BC concentrations being from 1800 to 2100 hours, nonSBC levels might be attributed to residential indoor emissions (e.g., cooking, burning candles). On the other hand, diesel traffic emission may be important sources of BC in NYC school environments (Patel et al. 2009). Fourth, we did not take into account minute ventilation differences in each environment (e.g., school vs. non-school vs. home) that could result in different doses of inhaled BC. Fifth, we were not able to control for other pro-inflammatory exposures, such as indoor allergens at schools or the impact of ventilation systems within the school. The lower ventilation rate often seen in schools is likely to have an inverse association with

BC and allergens brought to school (e.g., cat or dog dander) or generated within schools (e.g., mold) because decreasing outdoor air would tend to decrease outdoor-derived BC (e.g., traffic emission) in schools while increasing indoor allergen concentrations. Last, this study focused on urban minority children based on their high-risk asthma prevalence; therefore, our findings may not be generalized to nonminority populations. Minority children living in urban communities experience unique social, economic, and environmental factors that place them at greater risk that may not be experienced by other populations. Despite these limitations, our signal of an association between SBC and FeNO was quite robust. Our sensitivity analyses indicated that the association between school exposure and airway inflammation were independent of BC exposure in non-school environments and at residential homes. Furthermore, this study is strengthened by the use of repeat prospective measures of environmental air pollutants and FeNO over time (5 d and 6 months later), which allowed us to account for seasonal variations in both BC and FeNO measurements (Cornell et al. 2012; Jung et al. 2010).

To our knowledge, this is the first study to demonstrate that personal BC levels measured specifically at school was associated with increased FeNO among urban schoolchildren. The observation that nonasthmatic children who are seroatopic had a significant association between airway inflammation and SBC exposure suggests that atopic children could have increased sensitivity to SBC exposure. Our results provide rationale for interventions that target improved air quality in urban schools and classrooms.

Acknowledgments

The data sets generated during and analyzed during this study are available from the corresponding author on reasonable request.

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